IN THE SPECIFICATION

Replace paragraph 170 with the following rewritten paragraphs:

[170] Since DNA variants/SNPs in close physical proximity often show strong dependency relationships (i.e., linkage disequilibrium), it is determined if a group of DNA variants (SNP haplotypes) are inherited together, and determined if screening for only a portion of these SNPs will be sufficient for identifying the haplotype. Analysis of large regions of various chromosomes indicate that discrete haplotype blocks (of tens to hundreds of kilobases) are generally present, each with limited diversity punctuated by apparent sites of recombination. To find haplotypes, cDNA is sequenced and searched for combinations of sequence variations that are seen repeatedly in multiple individuals. Peripheral blood mononuclear cells (PBMC) were from 38 donors, and were stimulated *in vitro* with PHA (7.5 µg/ml) for 24 and 72 hours, or with Concavalin A (2µg/ml) for 24 hours. PMA (20ng/ml) and Ionomycin (1µM) were added during the last six hours of stimulation. The cells were then harvested and the total RNA was extracted using Trizol reagent(Invitrogen). To obtain cDNA templates for sequencing, RNA was reverse transcribed using Superscript II reverse transcriptase (Invitrogen), according to the manufacturer's protocol. The cDNA were used to PCR amplify the full length of *Tim* cDNA using Herculase Hot Start^{tm} high fidelity polymerase (Stratagene). The PCR primers were: ~~(SEQ ID NO:37)~~ (SEQ ID NO:41) GTGTCTGACAGTGGCGTA (forward), ~~(SEQ ID NO:38)~~ (SEQ ID NO:42) TTTGCCCAGGCAGAACCA (forward), (SEQ ID NO:43) CCACCCAAGGTCACGACT (reverse), ~~(SEQ ID NO:39)~~ (SEQ ID NO:44) ATGCCACGGACTAAGACC (reverse). The PCR products were purified with Qiagen QIAquick gel extraction reagents, and sequenced using four internal sequencing primers for *Tim1* and two internal sequencing primers for *Tim3*.

Replace paragraphs 202-203 with the following rewritten paragraphs:

[202] *Identification of Polymorphisms:* Peripheral blood mononuclear cells (PBMC) were obtained from 23 of these subjects, purified according to standard protocols and polyclonally activated with ConA or with PHA and PMA in vitro, prior to purification and reverse transcription of total RNA. The complete coding region of TIM-1 was amplified and sequenced with dye terminating methods at the Stanford Protein and Nucleic Acid facility, using the following primers for PCR with Herculase Hot Start Polymerase (Stratagene), ~~(SEQ ID NO:37)~~ (SEQ ID NO:45) 5'-GGAATTCGTCGACCACCATGCATCCTCAAGTGGTCATCTTA-3' and ~~(SEQ ID NO:38)~~ (SEQ ID NO:46) 5'-GGAATTCGCGGCCGCTCATTAGTCCGTGGCATAAACAGTATT-3', and for

sequencing, ~~(SEQ ID NO:39)~~ (SEQ ID NO:47) 5'- TCAAGTGGTCATCTTAAGCC-3', ~~(SEQ ID NO:40)~~ (SEQ ID NO:48) 5'-TAAACTCTCAAAG-AGCACCACT-3', ~~(SEQ ID NO:41)~~ (SEQ ID NO:49) 5'-ACAGACTCCAGCATAGATTCCT-3', ~~(SEQ ID NO:42)~~ (SEQ ID NO:50) 5'-GCACCAA GACAGAAATACAGAC-3', and ~~(SEQ ID NO:43)~~ (SEQ ID NO:51) 5'-AGAAGCACCCAAGACAGAAATACAGACTCCA-3'. The following nonsynonymous changes were identified by comparing our sequences to the NCBI reference sequence AF043724: 157insMTTTVP, 195delT, and A206T. To validate these polymorphisms, TIM-1 cDNA from five heterozygous donors was cloned into TOPO-TA sequencing vectors (Invitrogen) and sequenced. Genomic sequence was examined to confirm that the polymorphisms described are attributable to genomic polymorphisms within exons, not alternate splicing.

[203] *Genotyping:* Genomic DNA was purified from EDTA treated blood, according to standard protocols, and the eighteen base pair insertion/deletion variants of exon 4 were genotyped by SSLP analysis of length polymorphisms with the following set of intronic primers: ~~(SEQ ID NO:44)~~ (SEQ ID NO:52) 5'-TTCTAGCTGGGCAATGACC-3' and ~~(SEQ ID NO:45)~~ (SEQ ID NO:53) 5'-(FAM)-CCGCAGCTCCTCATTAGAAG -3'. Genotyping was performed using an ABI 3100 capillary electrophoresis sequencer with GeneScan software.